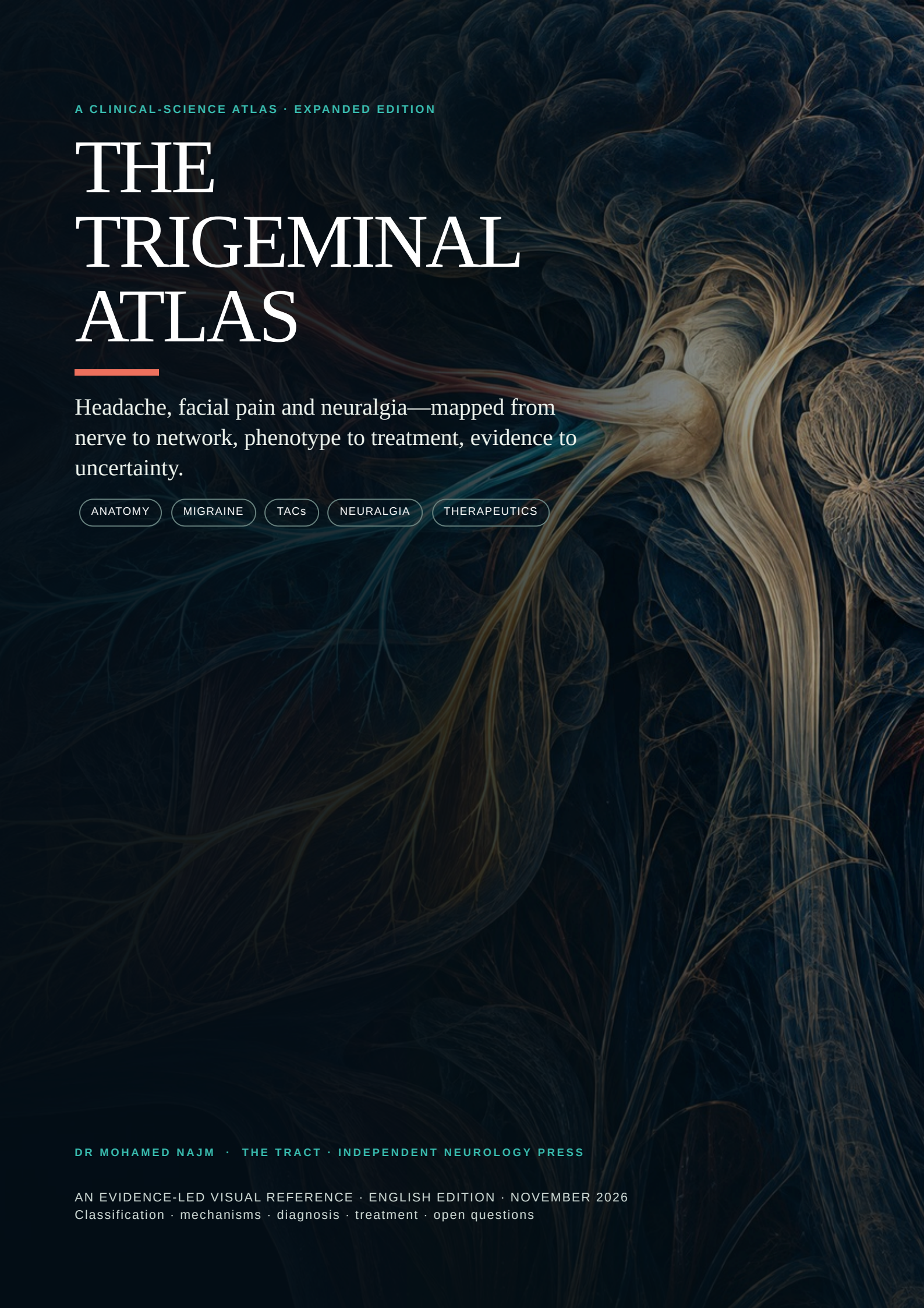
A CLINICAL-SCIENCE ATLAS · EXPANDED EDITION

# THE TRIGEMINAL ATLAS

Headache, facial pain and neuralgia—mapped from nerve to network, phenotype to treatment, evidence to uncertainty.

ANATOMY · MIGRAINE · TACs · NEURALGIA · THERAPEUTICS

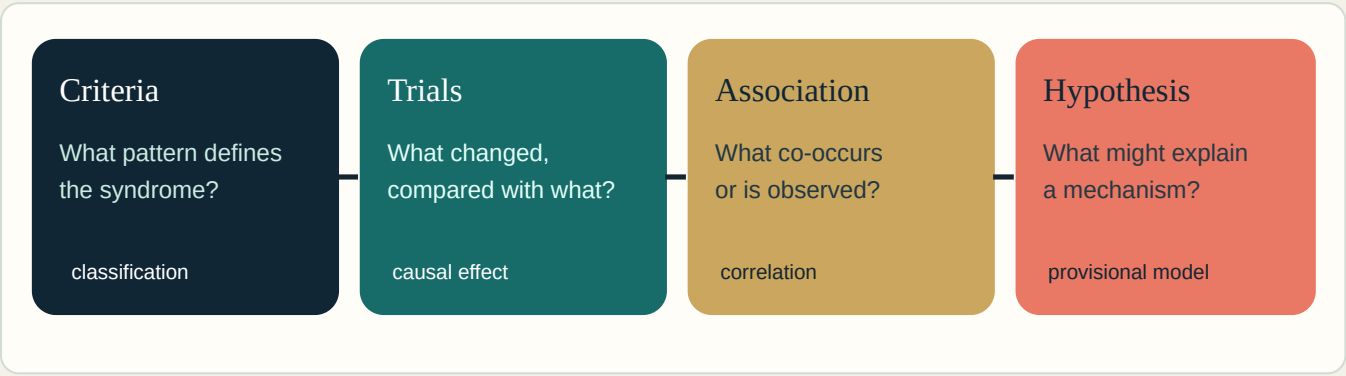


DR MOHAMED NAJM · THE TRACT · INDEPENDENT NEUROLOGY PRESS

AN EVIDENCE-LED VISUAL REFERENCE · ENGLISH EDITION · NOVEMBER 2026
Classification · mechanisms · diagnosis · treatment · open questions

# One nerve. Many clinical patterns.

The trigeminal system is a shared route, not a master diagnosis. The pattern of pain and the quality of evidence decide what the route means.




## Read by question

Start with time course, triggers, associated symptoms and sensory findings. Then review the nerve map. A shared anatomical pathway does not make migraine, cluster headache, trigeminal neuralgia (TN) and painful trigeminal neuropathy the same disease.

ICHD-3 criteria are classification rules. A stated duration or frequency is a practical diagnostic boundary, not a biological wall. People can have overlapping disorders, atypical presentations or secondary causes. [1]

## Read the evidence label

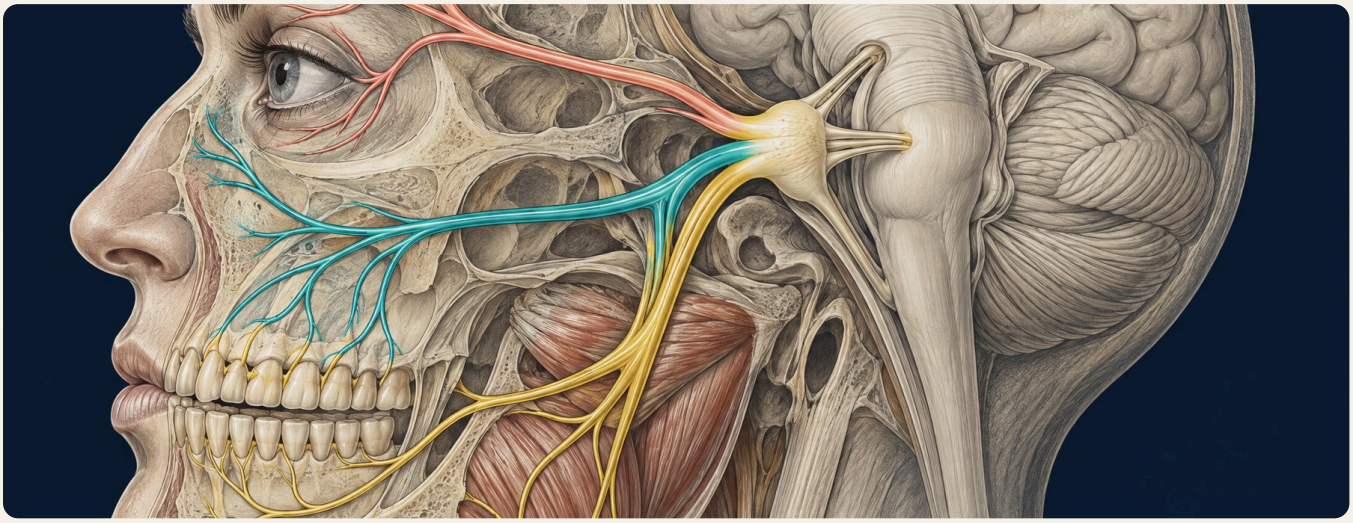
**Randomized trial:** estimates an intervention effect in a defined sample and time window. **Guideline:** combines evidence with clinical judgment and local priorities. **Imaging association:** shows a correlated signal, not necessarily a cause. **Hypothesis:** a model still being tested.

Percentages in this atlas retain their denominator and outcome. Results from different endpoints or study designs should not be compared as if they were a single contest.

> **The organizing principle:** identify the clinical phenotype first; interpret mechanism second; choose treatment by evidence, risk and context.

# Three divisions. One complex map.

Cranial nerve V
sensory + motor



## V1
### Ophthalmic
Forehead, upper eyelid, cornea and parts of the nose. Sensory division; its corneal afferents are clinically important.

## V2
### Maxillary
Midface, cheek, upper lip, palate and upper teeth. Sensory division.

## V3
### Mandibular
Lower face, lower teeth, and general sensation from the anterior tongue. Also carries motor fibers to muscles of mastication.

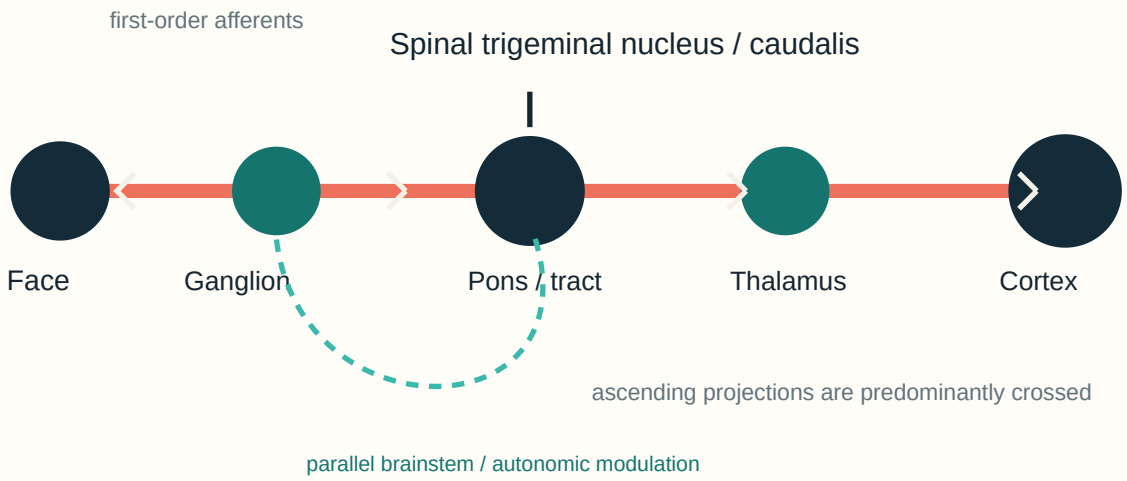
V1 forehead · eye → V2 midface · upper jaw → V3 lower face · jaw motor

The trigeminal ganglion houses the primary sensory cell bodies for most facial sensation. A notable exception is proprioception: primary sensory neuron cell bodies for jaw-muscle proprioception lie in the mesencephalic trigeminal nucleus. V3's mixed sensory-motor role is one reason facial and jaw complaints can involve sensation, chewing or both. [2,3]

> **Territory is not etiology.** Pain over a V2 region may come from tooth disease, sinus/eye disease, migraine, neuralgia, neuropathy or other causes. A face map guides examination; it cannot make the diagnosis by itself.

# How facial signals enter the brain.

Pain transmission is not a wire from a sore spot to a single “pain center.” It is a set of relays, descending pathways and interacting networks.




Facial nociceptive afferents enter the pons. Many travel caudally in the spinal trigeminal tract and synapse in the spinal trigeminal complex, especially its caudal region. Second-order projections ascend—predominantly across—to thalamic relays and cortical networks. Parallel brainstem circuits shape autonomic responses, arousal and affect. This simplified drawing leaves out multiple nuclei and bilateral connections. [2,3]

## Convergence

Touch, temperature and nociceptive inputs interact in trigeminal brainstem circuits. Their convergence is relevant to tactile triggers and sensitization, but does not mean every condition uses the same cellular pathway.

## Modulation

Brainstem and higher networks can amplify or dampen sensory input. The same incoming signal can be experienced differently across contexts, attacks and individuals.

# Mechanisms are a stack of models—not a single switch.

Headache science moved beyond the older idea that migraine is simply a dilated artery. Current models include peripheral trigeminal afferents, meninges, brainstem relays, hypothalamic and cortical systems, chemical mediators and sensitization. The diagrams below distinguish biological pathways from claims of causal sufficiency.

**HOW CONFIDENTLY CAN WE SAY IT?**

| Established clinical pattern | Therapeutically validated | Observed association | Open mechanism |
|---|---|---|---|
| classification criteria | target changes outcomes | co-varies with attacks | causal ordering uncertain |

These are different kinds of evidence, not a ranking of scientific value.

## Trigeminal–vascular signaling

Meningeal afferents and ascending trigeminovascular pathways provide a supported route for nociceptive signaling. This explains how meningeal inputs can engage the trigeminal system; it does not settle what initiates every migraine attack. [8]

## CGRP relevance

Human provocation studies and effective CGRP-targeting therapies support a role for CGRP in migraine. They do not show that CGRP is necessary or sufficient for all spontaneous attacks. [9–11]

## Central sensitization

Cutaneous allodynia and hypersensitivity can accompany migraine and other pain states. Peripheral and central sensitization are distinct concepts; clinical markers are indirect and causal roles in chronification remain unsettled. [8]

## What is not established

No single mechanism explains all phenotypes. A response to a medicine does not prove the disorder’s origin, and imaging activation is not proof that a region caused an attack.

> **Evidence discipline:** “involved in,” “associated with,” “can provoke,” and “causes” are not interchangeable verbs.

# Time, trigger, territory, companions.

A compact differential starts with the shape of an attack. Duration alone is never enough, but it is a powerful first clue.

| Pattern | Typical timing | Helpful accompanying features |
|---|---|---|
| **Migraine** | 4–72 h untreated in adults [4] | Nausea and/or photophobia + phonophobia; activity aggravation; aura in a subset |
| **Cluster** | 15–180 min | Same-side tearing/red eye/nasal symptoms and/or agitation |
| **Paroxysmal hemicrania** | 2–30 min | Frequent unilateral autonomic attacks; absolute indomethacin response in criteria |
| **SUNCT / SUNA** | 1–600 s | Very brief unilateral neuralgiform attacks with cranial autonomic signs |
| **Trigeminal neuralgia** | Fraction of a second–2 min | Shock-like paroxysms triggered by innocuous stimulation |
| **Painful trigeminal neuropathy** | Usually continuous / near-continuous | Burning/tingling pain; sensory dysfunction or nerve lesion may be present |

These are ICHD-3 descriptors, not a biological stopwatch. A series of TN paroxysms can feel like a prolonged bout; a person can have more than one headache disorder; and TN can include continuous interictal pain. Classify individual attack form and the overall clinical course separately. [1,4–7,17–19,23,24]

> **Ask in order:** When did it begin? How long is each burst? What provokes it? What occurs on the same side? Is sensation normal between attacks? Is the pattern new or changing?

# Migraine is a neurologic diagnosis.

For migraine without aura, ICHD-3 requires at least five attacks lasting 4–72 hours untreated or unsuccessfully treated. At least two of these pain features are present: unilateral location, pulsating quality, moderate/severe intensity, or aggravation by routine activity. The attack also includes nausea/vomiting or both photophobia and phonophobia. Unilateral pain is common but not mandatory. [4]

**Attack history** ≥5 episodes → **Duration** 4–72 h → **Pain features** ≥2 of 4 → **Associated symptoms** nausea or light + sound sensitivity

EPISODIC FREQUENCY

## Count headache days

Attack burden is not captured by severity alone. Record headache days, migraine-feature days, duration, acute medicines, function and recovery. A diary can make frequency and medication use visible across weeks.

CHRONIC MIGRAINE

## ≥15 days / month

Headache on ≥15 days/month for >3 months, with migraine features on ≥8 days/month, meets the ICHD-3 chronic migraine framework. Medication-overuse headache can coexist and is separately considered. [5]

## Aura is not “any visual symptom”

Typical aura consists of fully reversible visual, sensory and/or speech-language symptoms with characteristic development. NICE describes gradual development over at least 5 minutes and duration of 5–60 minutes. A new or atypical focal neurologic symptom should not automatically be assumed to be migraine aura. [6,30]

> **Clinical classification is not an imaging result.** There is no routine scan that “shows migraine.” Imaging decisions depend on the presentation, examination, stability and red flags; see the diagnostic pathway on page 19.

# A powerful model for aura—with important limits.




Hypothesized relation; not a fixed sequence in every attack.

Aura-associated cortical propagation: about 3–3.5 mm/min in human observations.

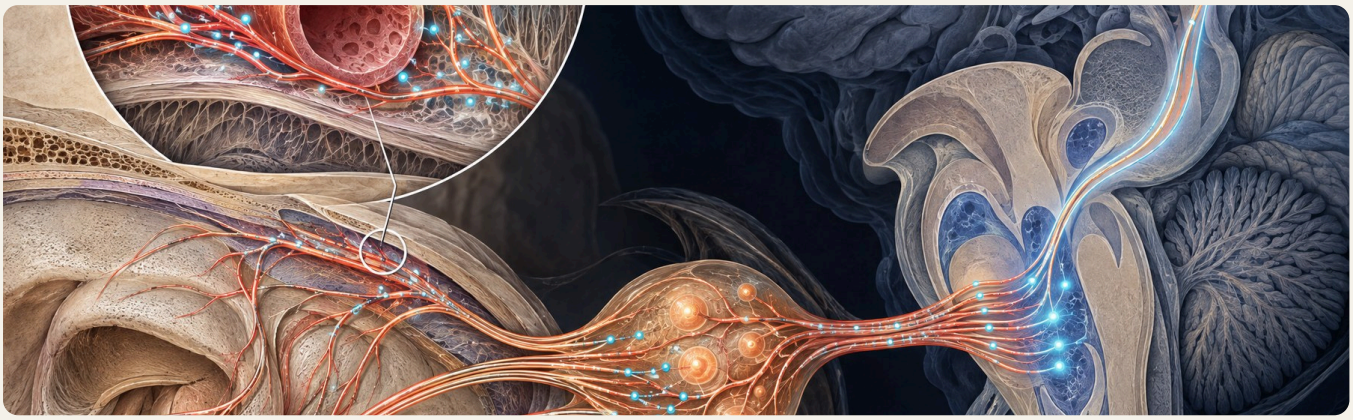
Cortical spreading depolarization (CSD)—a slowly propagating wave of altered cortical activity—is strongly associated with migraine aura. It is biologically plausible that such a cortical event can influence trigeminal nociceptive pathways; animal models support interactions. But CSD is not established as the initiator of all headache, nor as a universal explanation for migraine without aura. [7]

A 2025 intracranial EEG report recorded aura-associated propagating activity at about 3 mm/min in one patient monitored for epilepsy surgery. It offers rare direct human evidence, but a single case cannot establish how often this occurs or whether it causes the headache phase. [7]

MECHANISM STATUS

**Well supported:** CSD and aura association. **Open question:** whether/how CSD triggers headache in different migraine phenotypes. **Not supported:** treating one case or rate as a universal attack blueprint.

# A pathway, a target, and an unfinished theory.



**Meningeal input** (trigeminal afferents) → **Peptide signaling** (CGRP among mediators) → **Brainstem / thalamus** (ascending nociception) → **Sensory networks** (pain + symptoms)

The trigeminovascular framework links nociceptive fibers around intracranial meninges and vessels to trigeminal ganglion and central pain pathways. CGRP can be released from trigeminal sensory neurons and participates in signaling. Human provocation studies show that CGRP infusion can trigger migraine-like attacks in susceptible participants; this supports biological relevance, but does not prove CGRP is necessary or sufficient for every spontaneous attack. [8,9]

Therapeutic success with CGRP monoclonal antibodies and gepants validates the target. It does not mean migraine is simply “too much CGRP,” a vascular disorder, or a single-pathway disease. The initiating event, central/peripheral ordering, and how peptide signaling interacts with cortical and autonomic networks remain active questions. [10,11]

## Peripheral sensitization

Heightened responsiveness of peripheral nociceptive terminals is one candidate contributor to throbbing pain and mechanical sensitivity.

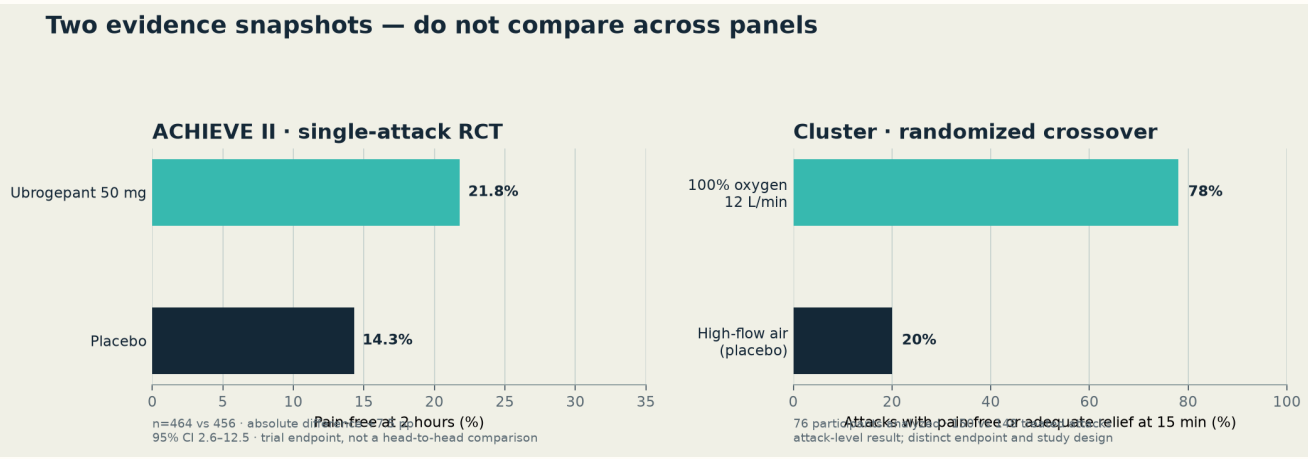
## Central sensitization

Amplified responsiveness within central circuits may help explain cutaneous allodynia and sensory gain. Human signs do not reveal every underlying cell mechanism.

**Don’t overread the vessel.** The presence of vessels near nerve fibers is anatomy. It does not establish that dilation is the sufficient cause of migraine pain.

# Treat the attack; measure the right outcome.

Acute therapies aim to reduce pain and associated symptoms, restore function and limit recurrence. Choice depends on attack features, previous response, contraindications, pregnancy status and medication-overuse risk. The 2025 American College of Physicians guideline recommends adding a triptan to an NSAID after inadequate response for moderate-to-severe acute episodic migraine in nonpregnant adults (strong recommendation); triptan plus acetaminophen is a conditional recommendation based on low-certainty evidence. [12]




Separate panels, separate disorders and endpoints. ACHIEVE II: single-attack, 2-hour pain freedom. Cluster oxygen trial: attack-level pain-free or adequate relief at 15 minutes. Do not compare across panels. [13,20]

In ACHIEVE II, a phase 3 single-attack study, ubrogepant 50 mg produced 2-hour pain freedom in 21.8% (101/464) versus 14.3% (65/456) with placebo: an absolute difference of 7.5 percentage points (95% CI 2.6–12.5). Absence of the most bothersome symptom was 38.9% versus 27.4%. These results apply to that trial population, dose, attack and timepoint; they do not predict an individual's response. [13]

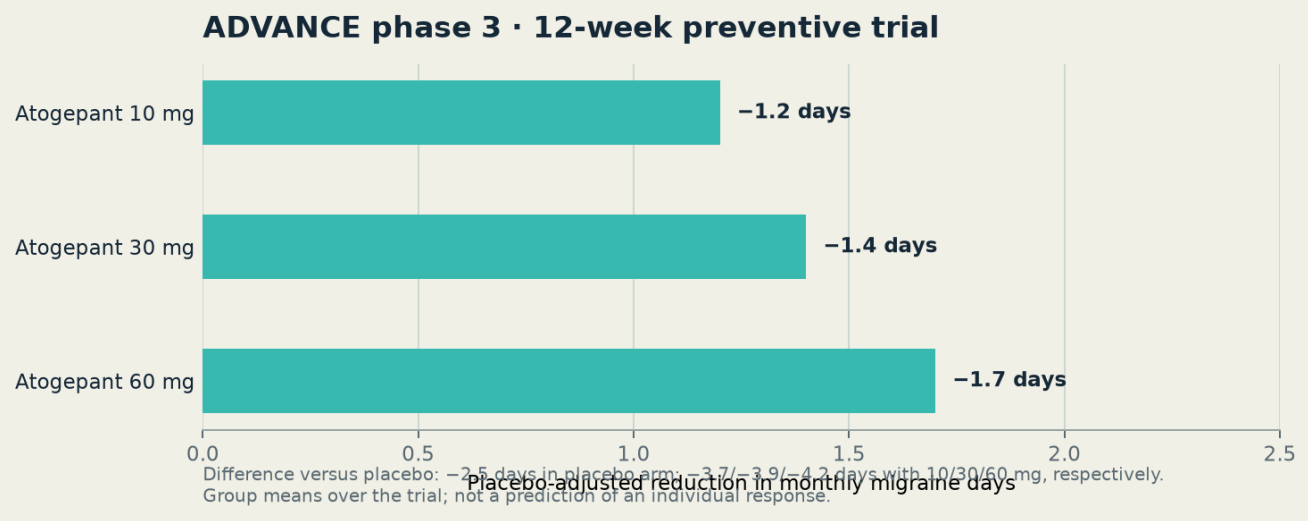
> **Outcome literacy:** "pain-free at two hours," "adequate relief at 15 minutes," and "days reduced per month" answer different questions. Relative and absolute differences, denominators, duration and comparator all matter.

For attacks with mild-to-moderate pain, an NSAID or acetaminophen may be considered when medically appropriate. For moderate-to-severe attacks, triptans remain an established acute option; antiemetics may be added when nausea is prominent, and non-oral routes can matter when vomiting or rapid escalation prevents reliable oral treatment. Gepants are acute options for some patients; triptan suitability, vascular history, drug interactions and pregnancy considerations need individual review. Opioids are generally avoided for routine migraine attack care because of limited role and potential harms. [12,30]

Medication-overuse headache is considered when headache occurs on at least 15 days/month with regular overuse for more than three months; thresholds differ by acute drug class. A diary that captures headache days and medication days supports more accurate assessment. [5,30]

# Prevention is a portfolio, not a single ladder.

Preventive treatment is considered when attack burden, disability, acute-medication use or patient priorities justify it. Traditional options include oral medicines selected by comorbidity and safety; onabotulinumtoxinA is used for chronic migraine in appropriate settings; CGRP-targeting monoclonal antibodies and gepants add mechanism-directed options. Local approvals, access rules and contraindications vary.




ADVANCE, 12-week phase 3 trial. Group-level placebo-adjusted mean differences, not predicted individual responses. [14]

In ADVANCE, mean monthly migraine days fell 2.5 with placebo and 3.7, 3.9 and 4.2 with atogepant 10, 30 and 60 mg, respectively; placebo-adjusted differences were −1.2, −1.4 and −1.7 days (all P<.001). The trial establishes efficacy over its period; it does not establish superiority to every preventive or long-term comparative safety. [14]

## CGRP-targeting therapies

The American Headache Society (2024) considers CGRP monoclonal antibodies and preventive gepants first-line prevention options without requiring failure of other classes. “First-line option” does not mean every patient should start there, or that head-to-head superiority is proven. [10]

## Access and context

NICE recommendations for rimegepant prevention use UK eligibility criteria, including prior preventive failures and reassessment. These are health-system access rules—not a global definition of efficacy. [15]

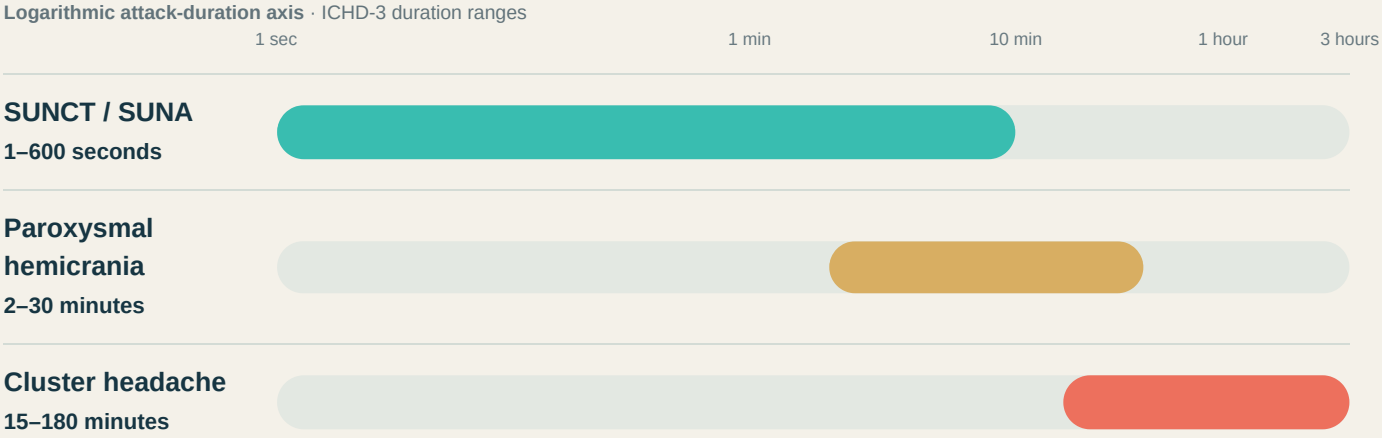
Established preventive classes include selected beta-blockers, topiramate, candesartan and amitriptyline, among others; their fit depends on comorbidities, adverse-effect profiles and reproductive considerations. OnabotulinumtoxinA is an evidence-based option for chronic migraine, studied in the PREEMPT program. Choice is not simply “old versus new”: tolerability, patient priorities, access and a measurable review plan matter. [16]

**Follow-up matters.** Agree on what outcome counts—fewer migraine days, shorter attacks, less disability or reduced rescue-medication use—and reassess benefit and harms after an appropriate trial period.

**Effect is not guaranteed.** Trial averages describe groups. Individual benefit, adverse effects, time to response and practical access differ. Treatment changes belong with a clinician.

# Different durations. Different signatures.

TACs typically combine unilateral trigeminal pain with ipsilateral cranial autonomic features, but are a family of distinct syndromes. The time bands below visualize ICHD-3 attack durations; frequency, autonomic signs, restlessness and indomethacin response also distinguish them. [17,18]




**Hemicrania continua:** continuous unilateral headache for more than 3 months; not a finite attack-duration interval and therefore not placed on this seconds-to-hours axis.

## Cluster headache

Severe/very severe orbital, supraorbital or temporal attacks lasting 15–180 minutes untreated; one every other day up to eight daily; ipsilateral autonomic signs and/or agitation. Episodic and chronic forms differ by remission pattern. [17]

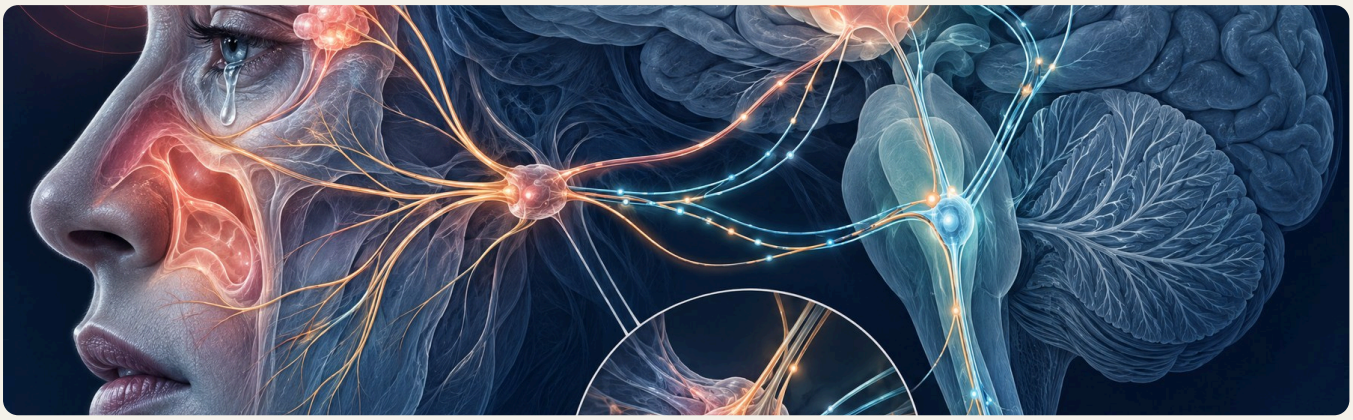
## Indomethacin-responsive TACs

Paroxysmal hemicrania and hemicrania continua require an absolute response to therapeutic-dose indomethacin in ICHD-3. This is a clinician-supervised classification feature, not a self-test or proof of mechanism. [18]

SUNCT includes both conjunctival injection and tearing; SUNA has only one or neither of those specific features. Very short attack duration does not imply mild disease: frequency and pain intensity can be substantial. [18]

**New side-locked autonomic pain deserves careful assessment.** A TAC-like phenotype may be primary or secondary; symptoms do not identify the cause on their own.

# Reflex, hypothalamus, distributed network.






A plausible circuit model for same-side tearing, eye redness and nasal symptoms.

The trigeminal–autonomic reflex provides a plausible, supported circuit explanation for ipsilateral tearing, conjunctival injection and nasal symptoms: trigeminal input can engage parasympathetic output via the superior salivatory nucleus, facial nerve and sphenopalatine ganglion. [21]

Hypothalamic-region activation in PET/fMRI studies, circadian/circannual regularity and melatonin observations support involvement of timing and distributed network systems—especially in cluster headache. Small samples and localization uncertainty mean these findings do not prove a single hypothalamic “attack generator.” Mechanistic evidence is concentrated in cluster and should not be generalized without qualification to every TAC subtype. [21]

> **Three evidence labels:** reflex circuitry—physiologically plausible; hypothalamic activation—imaging association; “the hypothalamus causes every attack”—not established.

# Three treatment clocks.

Cluster treatment is organized around speed: stop an attack, bridge a bout while preventive therapy takes effect, and reduce attacks across the bout. The European Academy of Neurology guideline (2023; evidence search through July 2022) strongly recommends 100% oxygen at ≥12 L/min for 15 minutes and 6 mg subcutaneous sumatriptan for acute attacks. [19]

**Acute**
minutes
oxygen / triptan

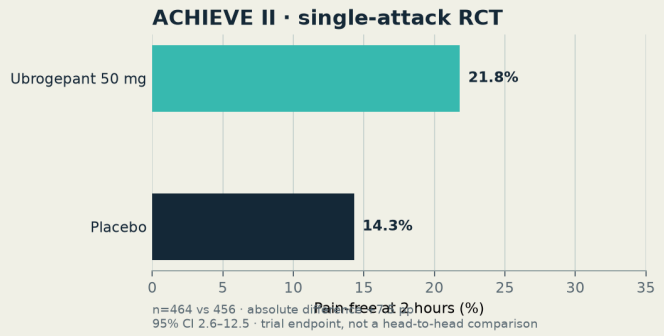
→

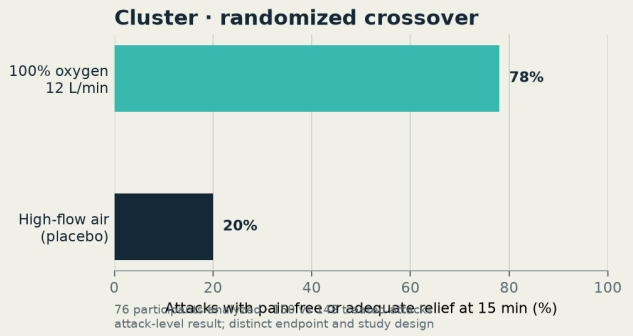
**Bridge**
days
selected steroid / GON block

→

**Prevent**
weeks
verapamil / selected options

**Two evidence snapshots — do not compare across panels**




Right panel: randomized crossover cluster trial. At 15 minutes, 100% oxygen at 12 L/min yielded pain-free or adequate relief in 78% of 150 treated attacks versus 20% of 148 attacks with high-flow air; 76 participants analyzed. Attack-level outcome, not participant-level success. [20]

## Prevention

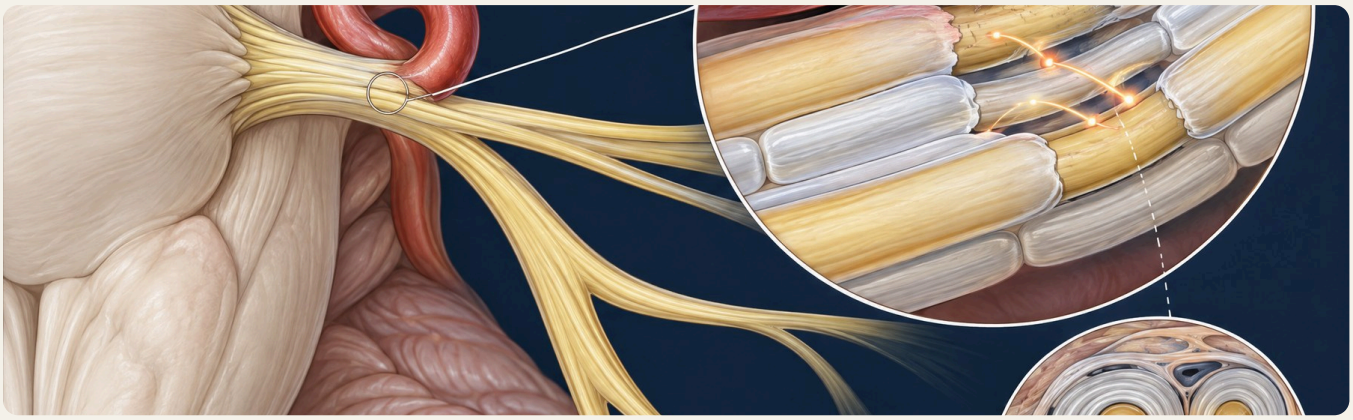
Verapamil is a standard preventive option; ECG monitoring is clinically important because conduction effects can occur. Corticosteroids and greater occipital nerve block are transitional approaches in selected cases. [19]

## Biologics & devices

Galcanezumab reduced attacks in episodic cluster in a trial that ended recruitment early; the evidence is not a basis to generalize to chronic cluster or other TACs. Noninvasive vagus nerve stimulation evidence differs between episodic and chronic cluster. [19,22]

**Acute oxygen is a treatment decision, not household oxygen advice.** Delivery device, flow, safety, contraindications and access require clinical planning.

# The shock has a clinical signature.



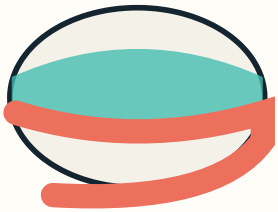
ICHD-3 describes recurrent unilateral pain in one or more trigeminal divisions: severe, electric-shock, shooting, stabbing or sharp paroxysms, typically triggered by innocuous stimuli, lasting a fraction of a second to two minutes. Some patients have concomitant continuous pain. [23]

Washing, brushing teeth, talking, chewing or a light touch may provoke a burst. There can be a refractory period after an attack. V2 and V3 are frequently involved; this distribution is descriptive, not a rule-out test for V1 or a diagnostic test by itself. [27]

CORE PHENOTYPE

## Paroxysm + trigger + territory

A brief neuralgiform attack in a trigeminal distribution is more informative than a generic report of “facial pain.” Continuous pain does not automatically rule out TN.



V2 cheek / upper jaw

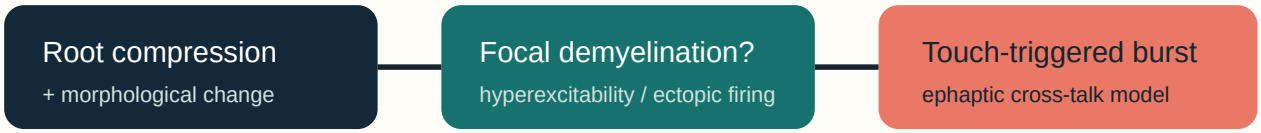
V3 lower jaw

Illustrative territories only

MRI is recommended during TN work-up because clinical features cannot reliably exclude secondary causes. The examination also helps assess neurovascular relationships for subtype classification and surgical planning. [25]

**Contact is not classical compression.** A vessel touching the nerve on a scan can be incidental. Classical TN requires neurovascular compression with morphological nerve-root change, such as displacement or atrophy. [23,25]

# Compression may matter—but does not explain all cases.




Leading explanatory model for some classical TN; not a complete proven account of every case.

Channel abnormalities, central sensitization and other contributors remain under study.

Focal demyelination near the root-entry zone, ectopic firing and ephaptic cross-excitation are a leading model for classical TN: a hyperexcitable fiber might allow tactile input to recruit a neighboring pain signal. Sodium-channel abnormalities and central sensitization are also discussed. These are explanatory models, not settled universal causes. [26,27]

## Medication first

NICE recommends carbamazepine initially. EAN recommends carbamazepine or oxcarbazepine as first-choice long-term medicines. Evidence for alternatives/add-ons—including lamotrigine, gabapentin, baclofen, pregabalin or botulinum toxin A—is less robust and context-dependent. [25,28]

## Evidence and safety

Carbamazepine has controlled-trial support, but the evidence base is largely small and short-term. It can cause rare serious skin reactions and blood dyscrasias; ancestry-related HLA screening and other monitoring depend on current product labeling and clinical setting. [28]

**When control is inadequate or medicines are poorly tolerated:** seek specialist review. Surgery is not a last-minute improvisation; it is a shared decision based on phenotype, MRI, health status and patient priorities. [25]

# Procedures solve different problems.

EAN recommends offering surgery when pain is inadequately controlled or medicines are poorly tolerated. For classical TN with morphological neurovascular compression, microvascular decompression (MVD) is the first-line surgical option in the guideline. If MRI shows no neurovascular contact, a neuroablative procedure may be considered. For idiopathic TN, evidence has not established a single best procedure. [25]

MVD

## Move the vessel

Open procedure intended to separate/compressing vessel from nerve while preserving the nerve. Requires suitable clinical/imaging context; entails operative risks and recovery.

PERCUTANEOUS LESIONING

## Interrupt transmission

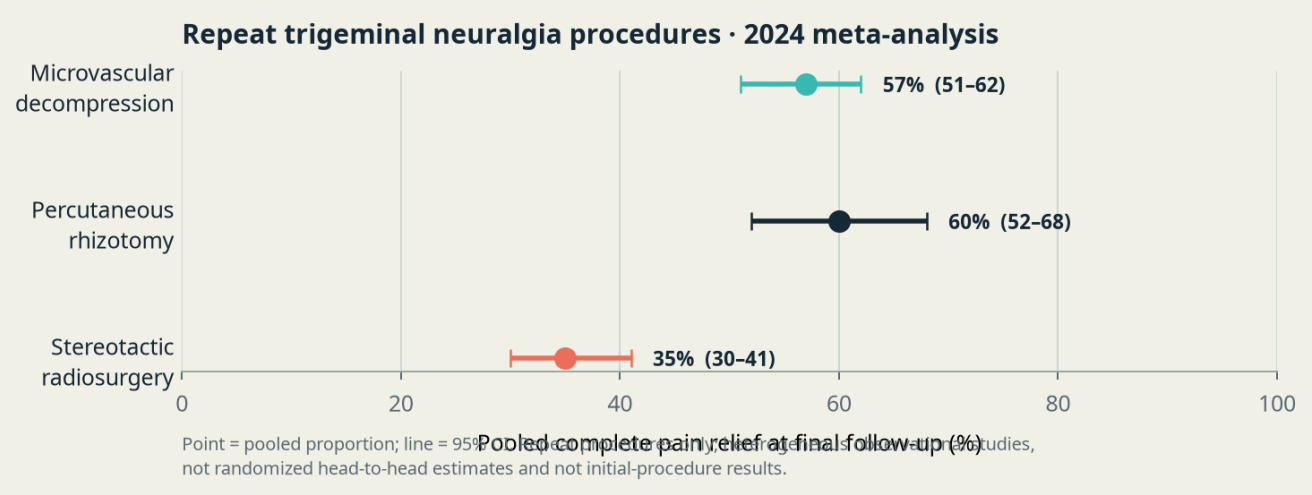
Procedures such as radiofrequency, balloon compression or glycerol intentionally injure/alter nerve transmission. Facial numbness and recurrence trade-offs matter.

STEREOTACTIC RADIOSURGERY

## Focused radiation

Non-incisional focused treatment; pain relief may be delayed, and sensory effects or recurrence are part of counseling.

A NOTE ON REPEAT SURGERY OUTCOMES




2024 meta-analysis of repeat procedures only. Observational, heterogeneous studies; not randomized head-to-head comparisons or first-procedure results. [29]

**Do not read this as a league table.** Patient selection, prior procedures, pain definitions, follow-up and study design differ. These repeat-surgery estimates do not predict an individual's result or describe initial surgery.

# Not every facial neuralgia is trigeminal neuralgia.

TRIGEMINAL NEURALGIA

## Paroxysmal pattern

Brief, severe, electric or stabbing bursts; innocuous trigger; trigeminal territory. Continuous concomitant pain can coexist.

PAINFUL TRIGEMINAL NEUROPATHY

## Often continuous

Burning, pins-and-needles or squeezing pain, often with sensory deficit, mechanical allodynia or cold hyperalgesia and an identifiable lesion/disease. [24]



V1–V3: trigeminal territories

V3 / oral territory: dental + jaw differentials

Throat / tonsil / ear: glossopharyngeal neuralgia

Back of scalp: occipital neuralgia / cervical referral

## Glossopharyngeal neuralgia

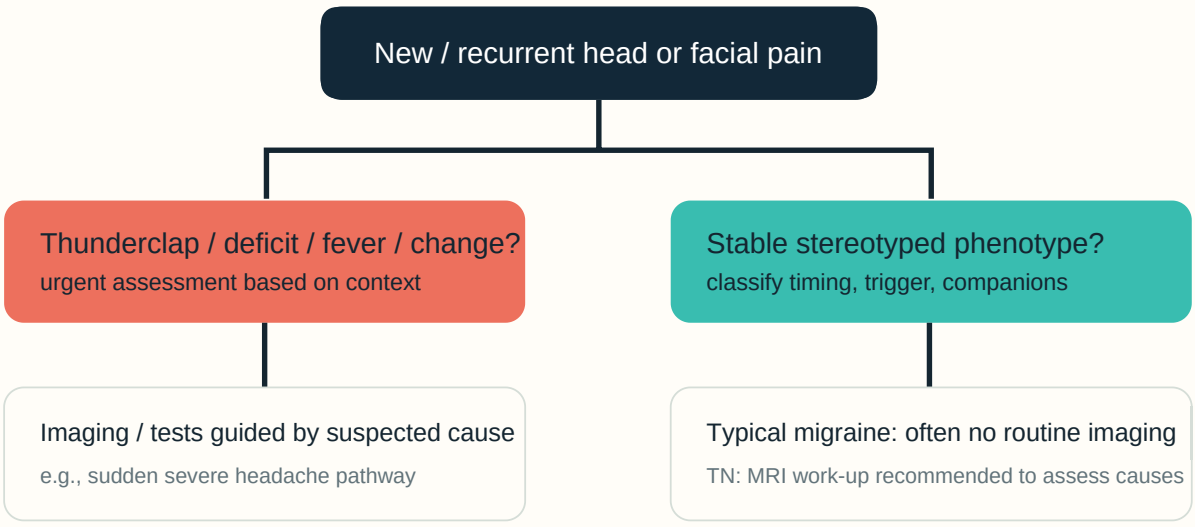
Brief unilateral pain in the ear, base of tongue, tonsillar region or beneath the jaw; swallowing, coughing, talking or yawning may trigger attacks. Rare vagal features include cough, hoarseness, syncope or bradycardia. [35]

## Occipital neuralgia

Paroxysmal pain in occipital nerve territories with local dysesthesia/allodynia, tenderness or trigger points; cervical joints and muscles can refer pain to the back of the head. [36]

Post-traumatic trigeminal neuropathy may follow dental, facial or other nerve injury. Sensory abnormalities are common in reported clinical series, but prevalence estimates vary by definitions and study design. The treatment evidence is not interchangeable with classic TN trials. [37]

# Investigate the right question.




A schematic aid, not a clinical decision rule.

NICE lists thunderclap onset (peak within five minutes), fever with worsening headache, new neurologic/cognitive/personality/consciousness change, recent trauma, cough/Valsalva/exertional or orthostatic headache, suspected giant-cell arteritis or acute narrow-angle glaucoma, and a substantial pattern change among features that can prompt investigation or referral. Context matters; these are not diagnoses. [30]

## Sudden severe headache

Noncontrast CT is commonly the initial imaging pathway. In a prospective cohort of neurologically intact adults scanned within six hours for acute headache peaking within an hour, CT detected all 121 SAHs (sensitivity 100%; 95% CI 97.0–100.0). Overall CT sensitivity was 92.9%; after six hours it was 85.7%. This conditional cohort result is not a universal guarantee. [32]

## Routine imaging is not universal

Stable typical migraine with normal examination and no red flags often does not need routine imaging. New/atypical TAC-like headache and suspected TN raise different imaging questions; EAN recommends MRI in all TN work-ups because symptoms cannot exclude secondary causes. [25,31,34]

SNNOOP10 is a structured prompt, not a validated rule-out score; the absence of listed features does not exclude every secondary cause. [33]

# What the field still cannot claim.

MIGRAINE

## What starts an attack?

How do cortical events, meningeal afferents, CGRP, brainstem and hypothalamic systems interact—and which order applies to which phenotype?

TACS

## Where is the generator?

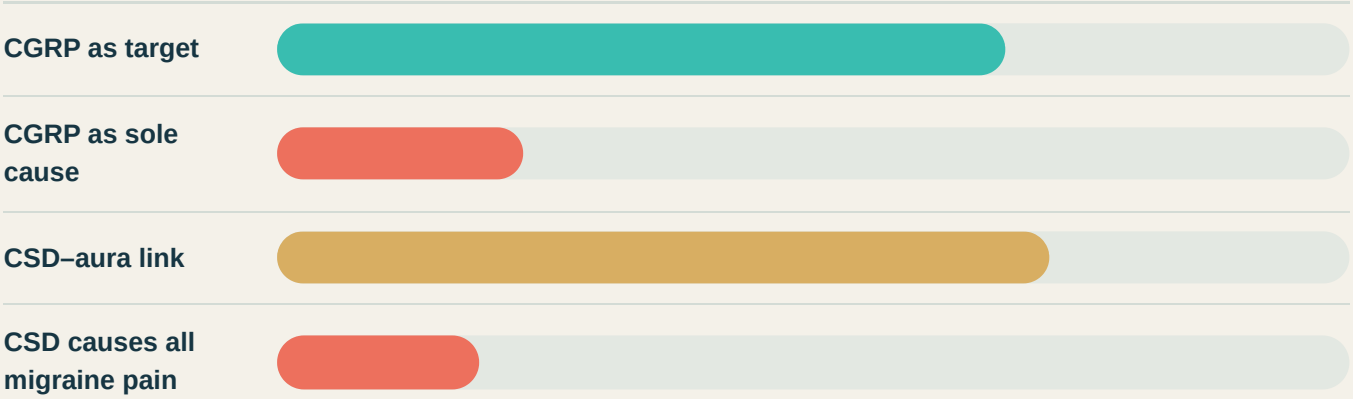
Imaging and circadian patterns implicate networks, especially in cluster. They do not establish a single anatomical switch or explain all TAC subtypes.

TN

## Why do some contacts hurt?

Morphological root change matters diagnostically, but the exact causes of pain, nerve hyperexcitability and treatment variation remain incomplete.

## Mechanisms that need careful labels




Bars are qualitative evidence labels for this article—not measured percentages or a formal certainty scale.

Clinical response can validate that a pathway is useful to target, but response alone rarely proves an upstream cause. Human provocation studies, imaging, sensory tests, animal models and randomized treatment trials answer different questions. Progress depends on linking phenotype, biology and outcome without collapsing those evidence types into one story.

> **What a global reference should do:** make mechanisms intelligible, make uncertainty visible, and make treatment claims traceable to a dated source and defined population.

# Seven questions worth carrying forward.

1. Where exactly?
2. How long per attack?
3. What trigger?
4. What accompanies it?
5. Normal sensation between attacks?
6. New or changing pattern?
7. Which evidence applies?

## Seconds + innocuous trigger

Raises a neuralgiform phenotype such as TN; evaluate trigeminal distribution, sensory findings and secondary causes.

## Hours + sensory accompaniments

Consider migraine criteria; count days and associated symptoms, not just side or pain quality.

## Unilateral pain + same-side autonomic signs

Consider TAC subtypes; duration, frequency, agitation and specific diagnostic features matter.

## Continuous pain + sensory change / injury

Consider painful neuropathy and its cause; do not automatically transfer TN evidence.

**Urgency overrides pattern matching.** Sudden maximal headache, new neurologic deficit, fever with worsening headache, altered consciousness, recent trauma or major pattern change calls for prompt clinical evaluation.

The trigeminal nerve is a powerful map of facial sensation and pain circuitry. It is not a shortcut around diagnosis. Recognize the pattern, locate the pathway, inspect the evidence, and keep uncertainty in view.

# Sources, with traceable claims.

[1] International Headache Society. ICHD-3 classification. ichd-3.org

[2] Terrier L-M et al. The trigeminal pathways. *J Neurol*. 2022. doi:10.1007/s00415-022-11002-4

[3] Kim HK et al. The Trigeminal Sensory System and Orofacial Pain. 2024. PMC11508441

[4] ICHD-3. Migraine without aura. Criteria

[5] ICHD-3. Chronic migraine. Criteria

[6] ICHD-3. Migraine with aura. Criteria

[7] McLeod GA et al. Mapping the migraine: intracranial recording of CSD in migraine with aura. Headache. 2025. doi:10.1111/head.14907

[8] Burstein R et al. Migraine: multiple processes, complex pathophysiology. *J Neurosci*. 2015. PMC4498450

[9] Lassen LH et al. CGRP may play a causative role in migraine. *Cephalalgia*. 2002. PMID 11993614

[10] Charles AC et al. CGRP-targeting therapies are a first-line option for prevention of migraine: AHS position statement update. 2024. PMID 38466028

[11] Sacco S et al. European Headache Federation guideline on CGRP monoclonal antibodies. 2022. PMC9188162

[12] Qaseem A et al. ACP guideline: acute episodic migraine in outpatient settings. 2025. Ann Intern Med

[13] Lipton RB et al. Ubrogepant for the treatment of migraine. ACHIEVE II. 2019. PMID 31742631

[14] Ailani J et al. Atogepant for the preventive treatment of migraine. ADVANCE. 2021. NEJM

[15] NICE. Rimegepant for preventing migraine, TA906. NICE

[16] Dodick DW et al. OnabotulinumtoxinA for chronic migraine: PREEMPT. 2010. PMID 20487038

[17] ICHD-3. Cluster headache. Criteria

[18] ICHD-3. PH, SUNCT/SUNA, hemicrania continua. TAC chapter

[19] May A et al. EAN guideline on treatment of cluster headache. 2023. PMID 37515405

[20] Cohen AS et al. High-flow oxygen for treatment of cluster headache: randomized crossover study. 2009. PMID 19996400

# Guidelines and further reading.

[21] May A et al. Trigeminal autonomic cephalalgias: mechanisms and treatment review. 2018. PMC5909131

[22] Goadsby PJ et al. Galcanezumab in episodic cluster headache. 2019. PMID 31291515

[23] ICHD-3. Classical trigeminal neuralgia. Criteria

[24] ICHD-3. Painful trigeminal neuropathy. Criteria

[25] Bendtsen L et al. EAN guideline on trigeminal neuralgia. 2019. doi:10.1111/ene.13950

[26] Bendtsen L et al. Advances in diagnosis, classification, pathophysiology and management of TN. *Lancet Neurol*. 2020. doi:10.1016/S1474-4422(20)30233-7

[27] Lambru G et al. Trigeminal neuralgia: a practical guide. 2021. PMC8461413

[28] Wiffen PJ et al. Carbamazepine for chronic neuropathic pain and fibromyalgia in adults. Cochrane. 2019. PMC6491112

[29] Repeat surgery for TN: systematic review and meta-analysis. 2024. World Neurosurg

[30] NICE. Headaches in over 12s: diagnosis and management, CG150; updated 2025. NICE

[31] American College of Radiology. Headache imaging appropriateness criteria. ACR

[32] Perry JJ et al. CT within six hours of headache onset to rule out subarachnoid hemorrhage. *BMJ*. 2011. doi:10.1136/bmj.d4277

[33] Do TP et al. Red and orange flags for secondary headaches: SNNOOP10. 2019. PMC6340385

[34] Evans RW et al. Neuroimaging for migraine: AHS systematic review and guideline. 2020. doi:10.1111/head.13720

[35] ICHD-3. Glossopharyngeal neuralgia. Criteria

[36] ICHD-3. Occipital neuralgia. Criteria

[37] Post-traumatic trigeminal neuropathy review. PMC11428572

[38] Lambru G et al. Medical treatment for SUNCT and SUNA: prospective open-label study. PMC7892380

**Scope:** This is a public-facing educational atlas. Diagnostic criteria do not replace assessment. Medicine options, contraindications, monitoring, approvals and coverage differ across people and jurisdictions. Recheck current labels and guidelines before clinical use.

# Made to be checked.

Every claim in this atlas points to a dated source. If something is wrong, out of date or unclear, tell us and it will be corrected in the next version.

## Author

**Dr Mohamed Najm** (Mohamed Najm Aldeen Alameen), neurologist in neurocritical care and neurological emergencies · FEBN.

ORCID 0009-0001-5506-2757

## This edition

The Tract Clinical Guides No. 7 · version 1.0 · November 2026.

Read online and download free at thetract.org/guides/trigeminal-atlas

## How to cite

Najm M. The Trigeminal Atlas: headache, facial pain and neuralgia — expanded edition. The Tract Clinical Guides No. 7, version 1.0. 2026. https://thetract.org/guides/trigeminal-atlas/

## How it was made

The ideas and direction are the author's. AI tools were used for literature search, drafting support and language editing. The author is responsible for the content. Our AI policy.

Illustrations are artistic renderings for orientation, not anatomical reference images.

## Licence and corrections

CC BY-NC-ND 4.0: share it unchanged, with credit, for non-commercial teaching.

Corrections and comments: contact@thetract.org

Version 1.0 for The Tract completes references 7 and 29 and resets two clipped labels on page 19.

**A teaching reference, not individual advice.** Diagnostic criteria support assessment; they do not replace it. Doses, approvals, monitoring and access differ between people and countries — check current labels and local guidance before acting on anything here.

The Tract · Independent Neurology Press

A free neurology library — no ads, no subscriptions, no sign-up. More clinical guides at thetract.org/guides